behind the counter in view of the customers. Wright's challenge is reportedly being backed the pro-smoking group FOREST, which sees it as a landmark which may prove that employers cannot impose smoking bans on staff. *See Press Association Newsfile*, March 25, 1993.

LEGAL ISSUES AND DEVELOPMENTS

UNITED KINGDOM

[54] "The Smokeless Zone," V. Harpwood, *Occupational Health Review*, March/April 1993

The author of this article examines the various legal options available to force employers to impose workplace smoking bans. It is conceded that the current legislative occupational safety and health framework will not support successful litigation. The author discusses the Veronica Bland case in which a settlement was reached between the Stockport Metropolitan Borough Council and one of its employees to resolve an allegation of injury due to ETS exposure and shows how it will not serve as legal precedent for other individuals who claim injury due to ETS exposure. The article concludes with a prediction that the pressure to change workplace smoking policies will come from the insurance industry which will likely respond to the first major award against an employer by demanding that companies impose smoking bans.

[55] Adoption Agencies Advised Not to Place Children With Smokers

On March 24, 1993, the British Agencies for Adoption and Fostering (BAAF) reportedly issued guidelines to adoption workers indicating that it is not in the best interests of children under the age of two to be placed with smokers. BAAF is the umbrella body for 180 fostering and adoption agencies. According to press reports, the guidelines do not have the force of law, but it is anticipated that BAAF's recommendations will be taken up by every agency in the country.

Citing statistics which purportedly relate smoking to increased risk of cot death, BAAF director Christine Hammond was quoted as saying, "If there is a choice between a smoking and a non-smoking family, it is hard to justify the extra risk of placing a child with the one that smokes." Some four or five couples are usually interested in adopting each of the 1,000 babies available for adoption every year. Smoking will join the list of other factors making families unsuitable for adoption, such as age, medical condition, substance abuse and household space.

Under-Secretary of State for Health Tim Yeo criticized the new policy and was quoted in press reports as saying, "It would be quite wrong for a child to be denied the chance of a loving home solely because there is a smoker in the household. There is no room for dogma or ideology in any aspect of adoption." *See The Independent*, March 25, 1993; *The Reuter Library Report*, March 24, 1993; *The Times*, March 25, 1993; *The Daily Telegraph*, March 25, 1993; *CNN* (Transcript), March 25, 1993.

OTHER DEVELOPMENTS

EUROPEAN COMMUNITY

[56] ◆Cigarette Confederation Publishes ETS Pamphlet

In March 1993, the Confederation of European Community Cigarette Manufacturers (CECCM) published a pamphlet which refutes many of the claims being made about ETS exposure and health. The document links complaints about indoor air quality to ventilation problems and criticizes the EPA Risk Assessment on ETS as "fancy statistical footwork."

UNITED KINGDOM

[57] ◆Symposium on Workplace Smoking Held in Glasgow

On March 10, 1993, a symposium on "Smoking in the Workplace" was held at the Royal College of Physicians and Surgeons of Glasgow. Some 100 delegates attended the event during which ASH proposals for imposing workplace smoking bans were presented as the optimal way of dealing with the issue. One speaker referred to the EPA Risk Assessment on ETS; another suggested that employers should consider

2046361357

Source: https://www.industrydocuments.ucsf.edu/docs/hqwl0000

a policy of employing only nonsmokers. Delegates were advised regarding the potential legal consequences of failing to protect employees from exposure to ETS.

[58] Union Employees Oppose Complete Smoking Ban

According to press reports, the Leeds Teaching Hospital Trust and its 5,000 union workers have been unable to reach agreement over a union demand for designated smoking areas. Evidently, the Trust abruptly ordered all employees to stop smoking at its facilities as of March 1, 1993. Although the union reportedly supports the policy, it says it is concerned that workers will "sneak away for a fag in a cubby hole and create a fire risk," said a union branch official. According to the chief executive of the Trust, segregated smoking areas have never worked and disciplinary measures will be taken against those who ignore the smoking ban. *See Yorkshire Evening Post*, March 1, 1993.

Meanwhile, hospitals and other health centres in Doncaster will reportedly ban smoking by staff beginning October 1, 1993. According to the chief executive of the Doncaster Healthcare Trust, staff members who persistently violate the rules could be dismissed. *See Doncaster Free Press*, March 1993.

[59] Schools Advised to Provide Smoke-free Environment

Schools in Camden have reportedly been advised that they should provide a smoke-free environment for their staff or they may be liable for prosecution. The advice follows the settlement in the Veronica Bland case which prompted a survey of Camden schools. Apparently, a number of the schools do not provide smoke-free staff rooms. *See Hampstead & Highgate Express*, March 1993.

[60] Pub Owners Refuse to Ban Smoking

Despite a national survey which reportedly found that most people do not want to be exposed to ETS when they go out for a drink, the majority of pub licensees are apparently not going to introduce smoking bans in their establishments. London pub company Wetherspoons, however, will reportedly introduce nonsmoking areas in all its outlets in the next month. According to the chairman of the Bath Licensed Victuallers' Association, it is up to individual pub landlords whether to introduce smoke-free areas in their premises. *See Bath & West Evening Chronicle*, March 1993; *The Star-Sheffield*, March 8, 1993.

In a related story, a cafe which has banned smoking was recently awarded the "Roy Castle Good Air Award." The award, named after an entertainer who claims ETS exposure caused his lung cancer, is apparently provided by the West Yorkshire Smoking and Health campaign and recognizes eating and drinking establishments that provide smoke-free areas. *See Huddersfield Examiner*, March 10, 1993.

Meanwhile, Roy Castle and fellow actor Richard Wilson have reportedly joined forces with the manufacturer of Nicorette nicotine gum and patches to educate the public about the purported dangers of ETS exposure. As part of the campaign, more than 12,000 pharmacies across the country will be stocking leaflets containing information about ETS. *See The Standard*, February 26, 1993.

[61] Study Links Child Snoring to ETS

According to the researchers at the Churchill Hospital Sleep Clinic in Oxford, children who snore are four times more likely to have a mother who smokes than nonsnorers. Enlarged tonsils are also apparently an important risk factor for snoring. In addition, the study linked aggressive and behavioral problems in children to parental smoking. *See The Oxford Times*, March 12, 1993.

World Airline News

[62] Bahrain

Gulf Air has reportedly announced it will ban smoking on all flights between Bahrain, Oman, Qatar and the United Arab Emirates. A spokesman for the airline says the ban will be in place by the end of March 1993. The spokesman said the measure is in line with directives from WHO on combating smoking. *See Moneyclips*, March 15, 1993.

[63] Iceland

Icelandair will reportedly ban smoking on all its European routes effective April 1, 1993. The airline apparently conducted a test ban and found that a "clear majority" favored nonsmoking flights. *See B.T.* (Danish Daily), March 7, 1993.

2046361358

Source: https://www.industrydocuments.ucsf.edu/docs/hqwl0000

[64] **Scandinavia**

Scandinavian Airlines System (SAS) says it will launch smoke-free European flights for a six-month trial period beginning on March 28, 1993. Flights with a maximum of two and one-half hours flying time, approximately 80 percent of SAS European flights, will be smoke free. Passengers will be polled during the trial period before a decision is made to make the bans permanent. *See Agence France Presse*, March 16, 1993. SAS provides air services in all Nordic countries.

[65] **Switzerland**

Crossair will reportedly introduce a smoking ban on flights which use SAAB 340 Cityliner or Fokker F50 aircraft. The ban, which will go into effect by the end of March 1993, will also apply to flights operated by Crossair on behalf of its parent company, Swissair. *See Neue Zuercher Zeitung*, February 25, 1993.

[66] **United Kingdom**

Cathay Pacific Airways has announced it will add the London-Hong Kong route to its list of flights where smoking will no longer be permitted beginning May 1, 1993. The route will join other international no-smoking routes, including those from Hong Kong to Australia, New Zealand, North America and routes within southeast Asia. An airline spokesman reportedly said the airline believes it will be in a position to be completely smoke free by 1995. *See Xinhua News Agency*, March 17, 1993.

MEDIA COVERAGE

AUSTRALIA

[67] **"Federal Court Warns Tobacco Institute Over Brochure," *The Australian Financial Review*, March 11, 1993**

This article highlights the warning Justice Sheppard gave TIA in its decision over outstanding matters in the lengthy litigation between AFCO and TIA. *See* issue 43 of this Report, March 19, 1993. The justice said that a TIA brochure being distributed to employers may have given a distorted impression of the federal court's decision in the case by omitting parts of the opinion in trying to persuade employers that ETS exposure is not a problem and its effects in the community have been overstated. The article also notes Justice Sheppard's criticism of a statement made during a radio interview by TIA executive director Jerome Mostyn to the effect that there is "no clear, conclusive evidence that passive smoking is injurious to the health of non-smokers." According to Justice Sheppard's opinion, there is no question about the ill effects of ETS exposure on asthmatics.

[68] **"Passive Smoking Danger Admitted," M. Date, *The Sydney Morning Herald*, February 26, 1993**

According to this article, the Tobacco Institute of Australia has admitted that ETS exposure might cause asthma attacks. The author quotes from a 32-page booklet on workplace smoking published by TIA as follows: "environmental tobacco smoke may temporarily produce or exacerbate symptoms in some asthmatics." Public affairs manager of the New South Wales Anti Cancer Council Stephen Woodward criticized the booklet and reportedly said that employers to whom it has been distributed described it as a joke. Woodward is quoted as saying, "If someone sues you, you cannot tender this document in court in your defence. It is not an expert opinion." The article contains other information from the booklet and concludes with TIA's assertion about the unresolved nature regarding the issue of safe doses for carcinogens.

UNITED KINGDOM

[69] ◆**"Passive Smoking Landmark," *Health and Safety Information Bulletin 207*, March 1993**

This article discusses the issues involved in the Veronica Bland case and observes that there is a dispute over whether the settlement will serve as precedent for other individuals claiming their respiratory problems are caused or exacerbated by workplace exposure to ETS. The Municipal Mutual Insurers, who reportedly advised Bland's employer to settle the case, has apparently claimed since then that the building in which Bland worked had severe ventilation, dust and dirt problems to which Bland was susceptible in addition to ETS.

According to a solicitor with the firm representing Bland, a principal issue in future cases may be the date

2046361359

Source: https://www.industrydocuments.ucsf.edu/docs/hqwl0000

by which employers should have taken action regarding workplace smoking. Local and health authorities, he speculates, due to their specialist knowledge, might be under a greater duty to act on ETS. The article also summarizes the EPA Risk Assessment on ETS and discusses UK employer smoking policies.

[70] "Smokers Forced Me To Quit Job, Says Clerk," A. Sambidge, *Western Daily Press*, February 20, 1993

According to this article, former civil servant Mark Langley was forced to quit his job as an administration officer at the Department of the Environment in Bristol, because he could no longer tolerate the smoking of his coworkers. Allegedly told by his personnel manager to put up with the smoke or quit, Langley is reportedly seeking legal advice about seeking compensation from his former employers.

[71] "Attitudes to Smoking," P. Madge, *The Safety & Health Practitioner*, March 1993

The author of this article, a nonsmoker who works as an independent consultant in legal liability and insurance, compares attitudes toward smoking in the United Kingdom, the United States and in South Africa. He reports the conclusion of the EPA Risk Assessment on ETS, and states that employers clearly have a duty to protect their employees from ETS exposure. The article concludes with advice to employers on gradually introducing workplace smoking policies in consultation with employees.

2046361360

Source: https://www.industrydocuments.ucsf.edu/docs/hqwl0000

APPENDIX A

The numbers assigned to the following article summaries correspond with the numbers assigned to the synopses of the articles in the text of this Report.

SCIENTIFIC/TECHNICAL ITEMS

LUNG CANCER

[28] **"Indoor Air Pollution and Lung Cancer in Guangzhou, People's Republic of China," Q. Liu, A.J. Sasco, E. Riboli, and M.X. Hu, *American Journal of Epidemiology* 137(2): 145-154, 1993**

"A case-control study comprising 224 male and 92 female incident lung cancer cases and the same number of individually matched hospital controls was conducted from June 1983 to June 1984 in Guangzhou, People's Republic of China, to evaluate the association between indoor air pollution and lung cancer risk."

"The goal of this study was to evaluate whether there is any relation between indoor air pollution resulting from domestic cooking practices and lung cancer occurrence."

"All cases and controls were interviewed in person. The interviewers obtained extensive information about the subject's general demographic characteristics, occupational history, history of respiratory diseases, family cancer history, smoking habits, spouse's smoking habits, cooking practices (including domestic fuel use), and residence history. After completing the questionnaire, the interviewer measured the size of the windows and doors that opened onto the outside of the building, thereby providing an estimation of ventilation capacity. The ventilation capacity of the kitchen was analyzed separately from that of the rest of the dwelling, hereafter designated the living area."

"Marital status, educational level, dialect, occupation, and living area did not differ significantly between cases and controls, although cases had a lower educational level and a smaller average living area than controls. Thus, we controlled for education, occupation, and living area when we analyzed other variables."

"As expected, the subjects with lung cancer showed increased frequencies of occupational exposure to hazardous working environments, a history of pulmonary tuberculosis, and a history of chronic bronchitis in both sex groups. When education, occupation, living area, and smoking were controlled for, the associations of lung cancer with the above risk factors were not substantially modified among men, but they were attenuated or even disappeared among women, except for exposure to dust. In contrast, the increased risk of lung cancer associated with a family history of cancer, found in univariate analysis, disappeared for both men and women when we controlled for the same variables. Smoking was strongly related in a dose-response manner to the risk of lung cancer in both men and women. . . . An increased risk of lung cancer was also observed among nonsmoking women who lived with a husband who smoked the equivalent of 20 or more cigarettes per day. [OR 2.9, 95% CI 1.2-7.3; for husband smoking 1-19 cigarettes per day, OR 0.7, 95% CI 0.23-2.2]."

"After adjustment for education, occupation, occupational exposure, pulmonary tuberculosis history, chronic bronchitis history, family cancer history, amount of smoking per day, and living area, as well as passive smoking (for women only), several variables pertaining to ventilation conditions were strongly associated with lung cancer risk. There was increased risk associated with having a window or door opening from the kitchen into the living area or bedroom, and for cooking in the living area or bedroom. For cooking in the living area or bedroom, the exposure odds ratio was 2.4 (95 percent CI 1.4-4.2) for men and 5.9 (95 percent CI 2.1-16.0) for women. Having windows or doors that opened in different directions so that indoor air could circulate also significantly influenced the risk of lung cancer. The relative risk for lung cancer tended to decrease with increasing size of ventilation openings in living areas and kitchens. For the best ventilated living area as compared with the least ventilated, the exposure odds ratio for lung cancer was reduced to only 0.14 (95 percent CI 0.04-0.51) for men and 0.02 (95 percent CI 0.00-0.21) for women. The exposure odds ratios for kitchen ventilation were 0.15 (95 percent CI 0.05-0.44) for men and 0.06 (95 percent CI 0.01-0.32) for women, respectively. The differences were statistically significant. A similar trend was found for the ceiling height throughout the apartment, but no clear trend was seen for the floor on which the subject lived."

"Passive smoking may also account for some excess risk, although increased risk was only observed in the women living with husbands who smoked heavily. No

2046361361

Source: https://www.industrydocuments.ucsf.edu/docs/hqwl0000

effect was seen for women married to light smokers. This may be explained by the reduced sample size and by the imprecise quantification of passive smoking. The results of other studies in Chinese women have suggested that passive smoking contributes to a slight increase in lung cancer risk."

"Since coal use, frequent home cooking, and lack of an apparatus for extracting fumes are universal in Guangzhou, it was difficult to find a significant difference between any population groups. However, this indicates that severe indoor air pollution exists for most families in Guangzhou, where people live in comparatively overcrowded conditions with poor ventilation. Better ventilation of houses could thus play a key role in improvement of the indoor microenvironment, and dissimilar ventilation conditions could be responsible for different exposure levels of lung cancer cases and controls. During the study period, there was in most houses no artificial ventilation such as air conditioning, so the indoor microenvironmental conditions depended mainly on natural ventilation. The area of ventilation (as defined by the area of openings to the outside) was a good representative measure of ventilation conditions. This is in agreement with other studies on indoor air pollution showing that the concentrations of pollutants are greatly affected by ventilation."

"In summary, the results of this study suggest that indoor air pollution produced during home cooking is a risk factor for lung cancer in Guangzhou, especially for women, who are more likely to be exposed to coal fumes and cooking oil vapors in the kitchen. This could contribute to the high rate of lung cancer in Chinese women. Further investigations are needed to clarify the precise nature of indoor air pollutants and their carcinogenic mechanisms. It would also be informative to conduct studies by major histologic type, particularly in Chinese women, among whom adenocarcinoma is unusually frequent."

Cardiovascular Issues

[29] "Environmental Tobacco Smoke and Coronary Heart Disease," A.K. Armitage, *Journal of Smoking-Related Disorders* 4(1): 27-36, 1993

"The debate regarding the association between smoking and cardiovascular diseases, especially coronary heart disease (CHD) has, during recent years, led to consideration of the possibility that exposure of non-smokers to environmental tobacco smoke (ETS) might increase the risk of such disease. The uncertainty about the existence of such an association is evident."

"Twelve epidemiology studies have now been published on which to base a judgment but few details were provided for two of the case-control studies and the studies of Martin et al., Palmer et al. and Butler have only been published as abstracts; full papers are still awaited."

"The 12 studies provided 15 different estimates of relative risk for either men or women, and 95% confidence intervals are available for all but two of these. 13 out of 15 indicate a relative risk of >1, but eight of these have a lower confidence limit of <1, indicative of a non-significant effect. Although the studies suggest an association, because most of the claimed relative risks lie between one and two, Hill's criterion of strength is not satisfied."

"Meta-analysis does not increase the *strength* of the association, although by reason of narrowing the 95% confidence intervals it may produce a mathematically, though not necessarily biologically, significant relative risk."

"Statistically, assuming no systematic bias, it would appear unlikely that 13 out of 15 estimates of relative risk >1 could have arisen by chance. Although some element of consistency amongst the published studies is indicated the possibility of publication bias may have accentuated the consistency of the association."

"Clearly, the amounts of nicotine and carbon monoxide absorbed from ETs are small and it is unlikely that either compound plays any role in the alleged increased relative risk for CHD of non-smokers exposed to ETS. It would be surprising if any other smoke component was absorbed in significant amounts from ETS."

"The case for ETS exposure causing CHD is wholly unconvincing because almost all of the accepted 'causational' criteria remain unsatisfied. Of particular concern is the weakness of the association, the likelihood of the existence of publication bias resulting in an overestimate of a very low relative risk, the lack of biological plausibility and the anecdotal nature of dosimetry assessment. At the present time, therefore, one is not able to conclude categorically that ETS is, or is not, harmful in a cardiovascular context. It is debatable whether the conduct of further epidemiologic studies, frequently recommended, is practical and justified. Small studies are a waste of time and money because at best they can only detect large risk as signifi-

2046361362

Source: https://www.industrydocuments.ucsf.edu/docs/hqwl0000

cant. Of course it is *theoretically* possible to conduct a prospective study, . . . which would control for all confounding variables and involve a sufficient number of subjects to provide reasonable statistical power. However, even large studies cannot distinguish with any certainty between a very low risk and no risk. Furthermore, without meaningful measurement of prolonged ETS exposure, results might still be inconclusive regarding the question of causation. In the meantime, it is wrong that an ETS/CHD health scare has been blown up out of all proportion in the last few years by a passionate anti-ETS health lobby. Interpretive opinions are not proven facts; they must be challenged and a more balanced point of view presented to the general public."

[30] **"Passive Smoking and the Risk of Acute Myocardial Infarction," C. La Vecchia, B. D'Avanzo, M.G. Franzosi, and G. Tognoni, *The Lancet* 341: 505-506, 1993**

"A possible relation between passive smoking and ischemic heart disease has been widely debated over the past decade, and at least 11 studies have been reported on the issue. . . . Most of these studies showed an increased risk, to a variable extent, of heart disease in never-smokers exposed to passive smoking. Even if the excess risk among non-smokers exposed to passive smoking were of the order of 20-30%, this would have a substantial impact on a public health scale, being responsible for a much larger number of deaths than that estimated for passive-smoking-related lung cancer deaths."

"A case-control study of acute myocardial infarction (AMI) was done in 1988-89 . . . on 916 first episodes of AMI and 1106 controls in hospital for acute diseases not related to any known or potential cardiovascular risk factor. From the original sample 113 cases of AMI and 225 controls admitted to the same network of hospitals for acute diseases not related to any known or potential cardiovascular risk factor were identified as currently married never-smokers. Exposure to passive smoking at home was investigated through questions on the spouse's habits, including smoking status (never-smoker, current, ex), number of cigarettes smoked per day, years the couple had lived together, and, if ex-smoker, time since quitting."

"Compared with subjects married to never-smokers, the multivariate relative risks, after simultaneous allowance for sex, age, education, coffee consumption, body mass index, serum cholesterol, hypertension, diabetes, and family history of AMI, were 0.91 (95% CI 0.36-2.28) for subjects married to an ex-smoker and 1.21 (0.57-2.52) for those married to a current smoker. Among subjects with a current smoker spouse, the RRs were 1.13 (0.45-2.82) for subjects with a spouse smoking less than 15 cigarettes per day, and 1.30 (0.50-3.40) for those with a spouse smoking 15 or more cigarettes per day."

"In this study, information on frequency of consumption of 14 selected indicator foods was available: no material differences were evident between subjects who lived with a smoker spouse and those who lived with a non-smoker. Average serum cholesterol concentrations were 207 and 209 mg/dL, respectively."

"The interpretation of this study remains inconclusive because of its lack of statistical significance, limited exposure assessment, and potential misclassification of the smoking status of subjects interviewed and their spouses. Still, these results, with the already accumulated evidence, are compatible with the hypothesis of a moderate effect of passive smoking on the risk of myocardial infarction."

Respiratory Diseases and Conditions — Children

[31] **"Relation of Passive Smoking as Assessed by Salivary Cotinine Concentration and Questionnaire to Spirometric Indices in Children," D.G. Cook, P.H. Whincup, O. Papacosta, D.P. Strachan, M.J. Jarvis, and A. Bryant, *Thorax* 48: 14-20, 1993**

"In this paper we examine the relation between spirometric indices and passive exposure to smoke in a large sample of children from 10 towns in England and Wales. In particular, we compare salivary cotinine concentrations with questionnaire measures of exposure in relation to spirometric indices in an age group, 5.5-7.9 years, where active smoking was unlikely to confound the issue. We measured mid and end expiratory slow rates (FEF_{50} and FEF_{75}) as well as forced vital capacity (FVC) and forced expiratory volume in one second (FEV_1) as there is some evidence that these are most affected."

2046361363

Source: https://www.industrydocuments.ucsf.edu/docs/hqwl0000

"Salivary Cotinine Concentration was strongly related to the number of smokers to whom the child was usually exposed. Undetectable cotinine was rare among those exposed to one or more smokers (18/1328 = 1.4%), whereas among those not regularly exposed there was only one reading above 14.7 ng/ml. Thirty five children had cotinine concentrations above 14.7 ng/ml."

"All spirometric indices show a tendency for children with higher cotinine concentration to have poorer ventilatory function. For all except the FEV_1:FVC ratio these differences were highly significant. The strongest association was with FEF_{50}, where each unit increase in cotinine concentration was associated with a fall of 14.3 ml/s."

"We ascertained exposure to cigarette smoke from four sources on the basis of the questionnaire: mother, father, others living in the same household, and anyone else looking after the child for more than two hours a week. The independent relationships of each of these sources of exposure to spirometric indices was examined by means of multiple regression analyses in which all sources were included simultaneously. All four sources of exposure tended to be associated with reduced spirometric measures. . . . Because the effects from the different sources of exposure seemed broadly similar (though few were individually significant) and because there was no clear evidence of dose-response effects with number of cigarettes smoked by mother or father we summarised exposure as the number of sources . . . For all indices except FVC there is a clear fall in respiratory function with increasing exposure. As with cotinine, the strongest association was with FEF_{50}, the estimated effect being a reduction of 51.0 ml/s for each additional source of exposure. In contrast to cotinine, exposure as assessed by questionnaire was negatively associated with FEV_1/FVC."

"We have shown the inverse associations between spirometric indices and both salivary cotinine and a questionnaire measure of passive exposure to smoking. . . . What is clear is that, in our data, a questionnaire score that combined several different sources of exposure was as good a predictor of flow rates as a single measurement of cotinine in young children."

"In our study there were no statistically significant associations with the smoking habit of the father and only a few spirometric indices were significantly associated with that of the mother. Using a composite score based on the number of sources of exposure, however, including one from outside the home, we found a clear relation between estimated degree of exposure to tobacco smoke and respiratory function. Nevertheless, the lack of a clear dose-response relationship with number of cigarettes smoked per day by the parents and the possibility that the mother's smoking is more important than the father's, which is supported by other studies, suggest that factors that modify exposure, such as the extent to which cigarettes are smoked in the presence of children, need to be taken into account. Our results also emphasize that questionnaire measures need to take account of other sources of exposure."

[32] **"The Decrease in Severity of Asthma in Children of Parents Who Smoke Since the Parents Have Been Exposing Them to Less Cigarette Smoke," A.B. Murray and B.J. Morrison,** ***Journal of Allergy and Clinical Immunology*** **91: 102-110, 1993**

"Since 1983 we have been gathering data on passive smoking and on asthma severity in children who have attended our clinic. From 1983 until 1986 we observed more severe asthma in children of smokers than in those of nonsmokers, but since 1986 there has been a marked lessening in the severity of asthma in children of smokers, their asthma being no more severe than that in children of nonsmokers. It appears likely that much of this improvement is the result of the parents' awareness of the harmful effects of smoke, and a consequent decrease in exposure of asthmatic children to smoke."

"Two sets of analyses were performed. The first was a preliminary one to determine whether parents were smoking fewer cigarettes and whether there had been a concomitant lessening in the severity of the children's asthma. After this a second analysis was carried out to assess if the change in asthma severity could be explained by the change in the parents' smoking habits. This was done by integrating the smoking and asthma severity data."

"The preliminary set of analyses showed that although there was little change in the total number of cigarettes the parents smoked per day when comparing the earlier and later periods, 17 versus 16 by mothers and 20 versus 19 by fathers, the mean number of cigarettes smoked when in the same room as the child

2046361364

Source: https://www.industrydocuments.ucsf.edu/docs/hqwl0000

decreased markedly, 7 versus 3 by mothers and 5 versus 2 by fathers. Concurrent with the lessened exposure to the mothers' smoke there was a marked improvement in the children's lung function."

"Three analyses of covarience were carried out for each of the four measures of asthma severity. . . . [S]mokers' children, compared with nonsmokers' children, had greater improvement in asthma score and in spirometric test results across the time period. . . . [T]his difference across the time periods in smokers' children is decreased if the number of cigarettes smoked when with the child is taken into account, as would be expected if exposure to the parents' cigarette smoke is a reason for the difference."

"Eighty percent of the parents who were questioned about their smoking after 1988 said that they had been told by their doctor not to expose their child to smoke, and that they attempted to avoid doing so by smoking outdoors or in another room or by using maneuvers such as smoking by an open window or blowing their smoke into the fire or into the exhaust fan in the kitchen."

"Not only was asthma less severe in smokers' children who were seen after July 1986, but also their lung function measurements were no longer lower than those found in nonsmokers' children. This large degree of improvement may be explained by smoke having caused the asthma, as well as aggravating asthma, in some smokers' children."

[33] **"Lung Function, Respiratory Illness, and Passive Smoking in British Primary School Children," R.J. Rona and S. Chinn, *Thorax* 48: 21-25, 1993**

"In the National Study of Health and Growth (NSHG), a nutritional surveillance system, data were collected on lung function from a substantial subsample of study areas in England and Scotland in 1987 and 1988. The sample included a range of socioeconomic, ethnic and geographical groups in the population. The aim of the analysis was to assess the contribution of respiratory illness and passive smoking to variation in lung function, with adjustment for potentially confounding social and biological factors. The factors associated with respiratory illness have been reported previously."

"Spirometry, in 2756 children aged 6.50-11.99 years, was carried out in a representative sample of English children, an inner city and ethnic minority sample, and a Scottish sample. Forced vital capacity (FVC), forced expiratory volume in one second (FEV_1), and forced expiratory flow rates of 25-75% and 75-85% (FEF_{25-75} and FEF_{75-85}) were measured and standardised scores obtained separately for the English representative sample, the Scottish sample and sub-groups in the inner city sample, white and Afro-Caribbean children and those originating from the Indian subcontinent. Multiple regression analyses were used to assess associations of FVC, FEV_1, FEF_{25-75} and FEF_{75-85} with the passive smoking and respiratory illness, with adjustment for a large number of potential confounders. Passive smoking was defined in terms of reported number of cigarettes smoked at home by each parent. The respiratory symptoms and illnesses assessed were wheeze, asthma and bronchitis attacks, cough in the morning, and cough at any other time as reported by parents."

"Inner city white parents smoked most heavily. There were very few children in the Asian groups exposed to smoking in the home of more than 15 cigarettes per day, as smoking is predominantly a male activity in these groups. Only 13 out of 681 Asian mothers were smokers. . . . For Afro-Caribbean children, maternal smoking was a more important component of total parental smoking because there was a larger percentage of one parent families. In the representative sample, Scottish parents smoked more than English parents."

"Most of the independent confounding variables in the analysis were not consistently related to all four measurements of lung function, or were not consistent in boys and girls. The only exceptions were birthweight, which was positively associated with most measures of lung function in both sexes, and study area in boys."

"The only significant associations detected were between maternal home smoking and reduced FEF_{25-75} and FEF_{75-85} in boys. Two other associations were of borderline significance in boys — FEV_1 with maternal home smoking, and FEF_{25-75} with total home smoking. Although not significantly different from zero, all other regression coefficients of lung function on maternal smoking were negative. In contrast, for paternal smoking at home most of the regression coefficients were positive. . . . In an analysis including both sexes, the interaction of sex and passive smoking was not significantly related to any of the measures of lung function. The differing effect of maternal and paternal smoking was confirmed by an analysis of both sexes."37

2046361365

Source: https://www.industrydocuments.ucsf.edu/docs/hqwl0000

"Afro-Caribbean children had lower lung function values than white children, whether living in inner city areas or from a representative sample, whereas children originating in the Indian subcontinent had lung function values intermediate between those of the Afro-Caribbean and the white children."

"We confirm that children whose parents report wheeze or asthma attacks in the child have significantly lower lung function test results than children without these symptoms. This study shows that a large range of potential confounding variables do not reduce the negative significant association between FEV_1, FEF_{25-75}, and FEF_{75-85} and symptoms of asthma."

"The associations between passive smoking and lung function were inconsistent. Our results are consistent with many reports which show that maternal and not paternal smoking is significantly associated with some measures of lung function."

"The amount of time spent by each parent with a child can explain the association of the child's lung function with maternal smoking but not paternal smoking. . . . In this study overcrowding was unrelated to lung function and number of children in the family was related only to FVC in girls, a measure not associated with passive smoking in our study."

"On the whole, our study illustrates the difficulties in making generalizations about the association between lung function and passive smoking in childhood. The effect of passive smoking on lung function is likely to depend on the amount of time spent by the smoker with the child, the number of cigarettes smoked, and the child's susceptibility. We have included a large number of confounding variables that could have explained a spurious relationship between lung function and passive smoking."

"In conclusion, this study has shown that parents' information about a child's asthma is associated with measures of lung function and that passive smoking can affect lung function. Close contact of the smoker with the child may be critical in damaging the growth of the lung. It must be recognised, however, that the mechanism of the association between a child's lung function and maternal smoking but not paternal smoking remains unexplained."

[34] **"Hispanic Children With Asthma: Morbidity," P.R. Wood, H.A. Hidalgo, T.J. Prihoda, and M.E. Kromer, *Pediatrics* 91: 62-69, 1993**

"A group of Hispanic children with moderate asthma followed in the clinics of the University of Texas Health Science Center at San Antonio were studied. Children aged 6 to 16 years with at least two acute-care visits or one hospitalization for asthma during the previous year were enrolled. Data sources included standardized questionnaires, spirometry, medical records, and school attendance records. Seventy-eight Hispanic children were enrolled in the study. Fifty-two (67%) of children had been hospitalized previously."

"The objectives of this cross-sectional study were to (1) describe the morbidity present in Hispanic children with asthma; (2) describe the knowledge, health practices, and perceived barriers to health care of these children and their families; and (3) identify factors that predict morbidity. This study is important because it provides detailed information about the needs and health status of a group of Hispanic children with asthma and identifies potential areas for intervention."

"The families of these children were poor and the majority of parents (62%) had not completed high school. Sixty-three families (81%) had an annual household income of $12000 or less. Fifty-five (71%) children had no health insurance and only 11 (14%) had Medicaid. Most children (78%) lived in two-parent households. Sixty-six households (85%) had four or more members."

"Almost all children took medications daily for asthma. The average number of medications that these children received for asthma was 2.8."

"[O]nly 45 (58%) [subjects] had three or more [spirometric] maneuvers that met ATS criteria for valid analysis. The spirometric data of these 45 children demonstrated mild to moderate airway obstruction, with a wide range of observed values."

"Children experienced a mean of 1.1 days of impairment per week, ie, days during which their sleep was disrupted by asthma symptoms or during which they were unable to participate fully in usual activities. They were absent from school an average of 13 days in the previous year."

2046361396

Source: https://www.industrydocuments.ucsf.edu/docs/hqwl0000

"Thirty-four children (44%) were exposed to cigarette smoke in the home. Twenty-five (41%) fathers and 14 (18%) mothers living in the home were smokers. Forty-eight (62%) parents identified cigarette smoke as a trigger for their child's asthma. Twenty-four (71%) of the 34 families with a smoker in the home identified cigarette smoke as a trigger for their child's asthma."

"This study documents the burden that asthma of moderate severity places on some Hispanic children and their families. We are aware that the results of this single study require replication and that several methodological issues need to be addressed before the results can be interpreted. First, this study was cross-sectional and was performed at a single site."

"Some of our findings may be attributable to problems associated with poverty."

"Second, . . . the findings cannot be generalized to other children with less severe illness."

"Third, although families were quite willing to participate if contacted, many families could not be contacted."

"Finally, the small sample size and relatively low reliability of some of the scales may have contributed to the small effects seen."

"This study reveals several potential targets for intervention. Two factors, knowledge about asthma and exposure to cigarette smoke in the home, were significantly associated with outcome in these children and therefore should be addressed in interventions."

"Passive exposure to cigarette smoke was a common finding and was significantly associated with impact of the illness on the family. Inasmuch as the success of smoking cessation interventions has been found to be related to both the number of sessions and the duration of contact with the program, the ongoing relationship of a physician with the parents of a child with asthma may a be good forum for smoking cessation advice. . . . At a minimum, physicians should encourage children with asthma and their families to limit the child's passive exposure to cigarette smoke."

OTHER HEALTH ISSUES

[35] **"Editorial: Give a Dog-End a Bad Name," A.D.S. Caldwell, *Journal of Smoking-Related Disorders* 41(1): 1-2, 1993**

"'Exposure to tobacco smoke and "well being and health" are incompatible. Discuss.' At first glance, this hypothetical examination question poses no great intellectual challenge. Cause and effect have been established beyond reasonable doubt, by means of well-designed pro- and retrospective epidemiological studies, between active cigarette smoking and a statistically significant increase in risk in bronchial carcinoma and coronary heart disease (CHD). . . . Serious questions have, however, recently been raised in this and other journals on the reliability of some data seemingly establishing some of these causal relationships."

"If data on active smoking are being occasionally called into question, there are even greater problems concerning passive smoking and environmental tobacco smoke (ETS). Cardiopulmonary disease, asthma, atherogenesis, lung cancer, leukaemia, retarded growth in children — in these and many more instances, a case has been made for ETS as a major aetiological factor. But assessing the impact of ETS is an exercise made hazardous by confounding variables lurking around every statistical corner. In the case of CHD, for example, some 300 risk factors have at some time or other been identified — by what means is it possible to unravel these data and point the finger with any degree of confidence at ETS *per se* as a major causative element?"

"In this issue, Dr. Armitage tackles the question of ETS and CHD, and his analysis of 12 major epidemiological studies leads him to conclude that the relationship between ETS and increased risk of CHD 'is not proven.'"

"The Journal of Smoking-Related Disorders is firmly behind all efforts to prevent the young from starting to smoke and convincing older people to stop. But we can also see the dangers inherent in overkill and the use of unsubstantiated generalisations. Campaigners are by nature evangelical in their approach — but the scientific argument has to be build on more solid foundations. To this end, the Editors of the JS-RD together with the publishers, Gardiner-Caldwell Communications (GCC Ltd), are actively canvassing support for an

2046361367

Source: https://www.industrydocuments.ucsf.edu/docs/hqwl0000

International Congress which will address some of the issues touched upon above. Anyone who would like further information should contact the Managing Editor directly at GCC Ltd, Macclesfield."

ETS Exposure and Monitoring

[36] **"Analysis of Tobacco-Specific N-Nitrosamines in Indoor Air," K.D. Brunnemann, J.E. Cox, and D. Hoffmann, *Carcinogenesis* 13(12): 2415-2418, 1992**

"A method was developed and applied for the assessment of tobacco-specific N-nitrosamines (TSNA) in indoor air polluted with tobacco smoke."

"Several tobacco-specific N-nitrosamines (TSNA) have been identified in tobacco and tobacco smoke. These include N-nitrosonornicotine (NNN), N-nitrosoanatabine (NAT) and 4-(methylnitrosamino)-1-(3-pyridyl)-1-butanone (NNK). . . . Both NNN and NNK are powerful carcinogens that induce benign and malignant tumors of the lung, nasal cavity, esophagus, pancreas and/or liver in mice, rats and hamsters. Recently it was suggested that TSNA act as causative agents for lung cancer in active smokers. It was the goal of this study to assess the contribution of TSNA to indoor air pollution."

"TSNA levels [were] obtained under different conditions in the test laboratory in which one, two and four cigarettes per 30 min were machine smoked simultaneously. . . . It is noteworthy that the NNK levels are [about] 10 times higher than those of NNN. . . . TSNA values [were] obtained at different indoor sites polluted with environmental tobacco smoke (ETS). The number of cigarettes smoked during the sampling time (3-8 h) was estimated to the best of our ability. NNN levels varied from not detected to 22.8 pg/l while NAT ranged from not detected to 9.5 pg/l. NNK levels ranged from 1.4 to 29.3 pg/l. . . . Assuming a respiratory rate of 10 l/min, a non-smoker exposed to these concentrations of TSNA may inhale up to 41 ng of NNN and up to 43 ng of NNK in 3 h. Taking as an average value of NNN and NNK in the mainstream smoke of a filter cigarette 360 and 130 ng/cigarette respectively, this would mean an exposure to NNN of 0.1 and to NNK of 0.3 cigarette equivalents."

[37] **"Relationship Between Environmental Tobacco Smoke Exposure and Carcinogen-Hemoglobin Adduct Levels in Nonsmokers," S.K. Hammond, J. Coughlin, P.H. Gann, M. Paul, K. Taghizadeh, P.L. Skipper, and S.R. Tannenbaum, *Journal of the National Cancer Institute* 85(6): 474-478, 1993**

"Environmental tobacco smoke contains more than 20 identified carcinogens to which nonsmokers may be exposed and is the only known environmental source of one of these carcinogens, 4-aminobiphenyl (4-ABP). 4-ABP was found to be a potent bladder carcinogen in workers in the dye industry, where its use has been discontinued for decades. The emissions of 4-ABP are more than 30 times greater in sidestream smoke than in mainstream smoke; therefore, passive smokers might receive substantial doses of this carcinogen. 4-ABP bonds covalently with hemoglobin, and this adduct might serve as a surrogate marker for a biologically effective dose."

"In a previous study, we reported finding 4-ABP-hemoglobin adducts in the cord blood of babies born to both nonsmokers and smokers. This observation indicates that 4-ABP or its metabolite crosses the human placenta and binds to fetal hemoglobin. The present study examines the relationship between quantitative measures of exposure to environmental tobacco smoke and the levels of 4-ABP-hemoglobin adducts in the same nonsmoking pregnant women reported in the previous study and compares these levels of 4-ABP-hemoglobin adducts to the adduct concentrations found among the same smoking pregnant women reported in our previous study."

"We measured exposure to environmental tobacco smoke by using the following three tools that have been described in detail previously: 1) At enrollment, the subject was asked to complete a detailed questionnaire, administered by an investigator, on passive smoking exposure during an average week and was asked a subset of questions on two later occasions to evaluate how exposures had changed; 2) the subject was asked to complete a 7-day diary of environmental tobacco smoke exposure; and 3) during the same week that the subject was completing the diary, the subject was asked to wear a passive monitor to sample the air for environmental tobacco smoke."

2046361368

Source: https://www.industrydocuments.ucsf.edu/docs/hqwl0000

"Fifty-four pregnant nonsmokers and 20 pregnant smokers were enrolled in this study. . . . Blood was collected and analyzed for hemoglobin adducts of 4-ABP for 40 of the nonsmokers and 15 of the smokers. The mean level of adducts among the 15 smokers was 184 pg of 4-ABP per gram of hemoglobin, while that for the 40 nonsmokers was 22 pg of 4-ABP per gram of hemoglobin. . . . In this study and in previous studies, there was no overlap between the values found for smokers and nonsmokers. The actual levels found in any population will depend on the smoking habits of smokers and the passive smoking exposures of nonsmokers, as well as on individual metabolic differences."

"To examine whether there was any relationship between the environmental tobacco smoke exposures and the levels of 4-ABP-hemoglobin adducts in nonsmokers, we categorized average nicotine exposure in the third trimester into low, moderate, and high environmental tobacco smoke exposure groups and calculated the median and mean adduct levels in each category. . . . As environmental tobacco smoke exposure increased, so did both the median and the mean 4-ABP-hemoglobin adduct levels. . . . The relationship between environmental tobacco smoke exposure and adduct levels was stronger in this study than in other studies, probably because we used more complete and objective measures of exposure."

"A statistically significant relationship was found between the weekly average exposure to environmental tobacco smoke during the third trimester of pregnancy and the levels of 4-ABP-hemoglobin adducts found at the time of delivery. This relationship remained after subjects with possible to gas stoves, a possible interference, were removed from the dataset. This result indicates that exposure to environmental tobacco smoke is related to levels of a known human carcinogen in nonsmokers. The increase is not dramatic, and the public health significance is unclear. The following facts, however, indicate that nonsmokers may receive a nontrivial dose of carcinogens from environmental tobacco smoke: 1) The carcinogen 4-ABP was detected in passive smokers at 14% the level found in smokers; 2) this ratio is consistent with the emissions of mainstream smoke and of sidestream smoke; and 3) greater amounts of other carcinogens, e.g., nitrosamines, are also emitted in sidestream smoke than in mainstream smoke. Although bladder cancer among passive smokers has not been studied, the incidence of lung cancer has been reported to be elevated among nonsmokers married to smokers. The relationship between environmental tobacco smoke exposure and 4-ABP-hemoglobin adduct levels contributes to the plausibility of epidemiologic evidence that environmental tobacco smoke is carcinogenic to passive smokers."

[38] **"Environmental Tobacco Smoke," A. Rodgman, *Regulatory Toxicology and Pharmacology* 16: 223-244, 1992**

"The U.S. Environmental Protection Agency (EPA) issued two draft documents in which it defined ETS as a carcinogen and designated it as a 'Group A carcinogen' in an effort to encourage regulation of smoking in the workplace."

"Of prime concern to the EPA were 43 smoke and tobacco components which, in one biological system or other and at doses far in excess of those encountered in MS or SS, had been reported as 'tumorigenic.' The EPA assessed the health consequences with regard to lung cancer of the listed 43 components as follows: 'Of the 99 compounds in tobacco smoke that have been studied in detail, at least 43 are complete carcinogens, each able on its own to cause the development of cancer in humans or animals.' This assessment is incorrect since most have not been shown to be (1) tumorigenic to any human tissue or (2) tumorigenic to lung tissue in laboratory animals. . . . In addition, the few that have produced lung tumors in laboratory animals have do so at dose levels far in excess of those encountered in MS, SS, or ETS."

"As a matter of Agency policy, the EPA overlooks the fact that progressive reduction of the dose of an administered material which produced a given effect in a bioassay eventually leads to a level at which no effect is observed. Despite much data to contradict it, the theory to which the EPA subscribes is that the only safe threshold limit value for a material tumorigenic in animals is a 'zero' dose for humans."

"When there is evidence to the contrary that a threshold does indeed exist for a compound under investigation, the EPA is required to consider such evidence and not adhere to the presumption of 'no threshold.' This article presents examples of such 'evidence to the contrary.'"

2046361369

Source: https://www.industrydocuments.ucsf.edu/docs/hqwl0000

"The failure to produce in MS-exposed laboratory animals the tumor type reported to be associated with smoking in humans is important not only with regards to the biological properties of MS itself but also with respect to that of diluted MS delivered to the caged animals. Early experiments involving 'whole body' exposure to diluted MS closely resemble ETS exposure, albeit at levels substantially greater than those usually encountered with ETS."

"If . . . these smoke-inhalation experiments more closely resembled passive smoke (or ETS) exposure than human exposure during actual smoking, then substantial evidence is available to demonstrate that exposure to 'passive smoke' (or ETS), more concentrated than that encountered in the human situation, is ineffective in induction of the tumor type supposedly associated with cigarette smoking in humans: Whole body exposure studies . . . were designed to determine the effect of MS on laboratory animals, with emphasis on lung cancer induction. Because of the whole body exposure, results are relevant to the evaluation of the biological effects of exposure to concentrated passive smoke. None of these exposures to concentrated passive smoke induced squamous cell carcinoma in the test animals."

"Comparison of the list of the 4800 or so identified components in tobacco smoke with various lists of compounds shown to possess inhibitory or anticarcinogenic action in carcinogenesis-type experiments with laboratory animals reveals not only that tobacco smoke contains numerous anticarcinogens but also that their levels in smoke far exceed those of the tumorigens in 'The List of 43.'"

"ETS is neither MS nor SS. Chemically, MS, SS, and ETS are qualitatively the same but quantitatively different; physically, they differ significantly."

"In active smokers, percentage retentions of MS PP [particulate phase], as measured by PP weight loss, range from 50 to 90%, values higher than would be predicted from particle size alone due to coagulation, electrical charge, growth by water condensation, evaporative transfer, and cloud effects with the latter two being the most significant. For ETS, the percentage retention theoretically should be 10-20%; a measured value of 11% has been reported."

"Only a few components on 'The List of 43' have been tested experimentally for tumorigenicity to lung tissue via inhalation. . . . It is doubtful that any of the 43 components should be included in 'The List of 43.' Examination of information on Hoffmann and Hecht's 43 listed components reveals that it is inappropriate to extrapolate from such a list to a relationship between MS exposure and lung cancer induction in smokers or between ETS exposure and lung cancer induction in nonsmokers."

"Fairness of public policy requires consideration of all available evidence, especially the evidence in the form of objective scientific data. Selective use of data to support conjectures that, in turn, support preconceived agenda will not lead to defensible policies, and it is certainly reprehensible science. The evidence presented here, if objectively examined by the EPA, should significantly influence its assessment of the ETS issue."

Indoor Air Quality

[39] **"The Effect of Varying Levels of Outdoor-Air Supply on the Symptoms of Sick Building Syndrome," R. Menzies, R. Tamblyn, J.P. Farant, J. Hanley, F. Nunes, and R. Tamblyn,** ***The New England Journal of Medicine*** **328(12): 821-827, 1993**

"This study was undertaken to test the hypothesis that symptoms of sick building syndrome could be reduced in mechanically ventilated office buildings by increasing the supply of outdoor air from 20 to 50 ft^3 (1.4 m^3) per minute per person."

"A randomized double-blind multiple-crossover trial was conducted to estimate the effect of changes in the outdoor-air supply in four office buildings. During three consecutive two-week blocks of time, building ventilation systems were manipulated to deliver an expected 20 or 50 ft^3 of outdoor air per minute per person to the indoor environment, corresponding to indoor carbon dioxide concentrations of 1000 or 600 ppm. Within each two-week block, the ventilation level was increased for one week and decreased for the other."

"Of 1838 eligible workers in the four buildings, 1546 (84 percent) participated in the study. The supply of outdoor air averaged 7 percent and 32 percent in the ventilation systems and 30 and 64 ft^3 (0.85 and 1.8 m^3) per minute per person in the work sites at the lower and higher ventilation levels, respectively. These changes in the

2046361370

Source: https://www.industrydocuments.ucsf.edu/docs/hqwl0000

supply of outdoor air were not associated with changes in the participants' ratings of the office environment or in symptom frequency (crude odds ratio, 1.0; 95 percent confidence interval, 0.9 to 1.1). After work-site measures of ventilation, temperature, humidity, and air velocity were included in the regression analysis, the adjusted odds ratio was also 1.0 (95 percent confidence interval, 0.8 to 1.2)."

"On average, a little more than half the participants reported at least one symptom each week, and the proportion reporting symptoms within each two-week block was virtually identical at the two levels of ventilation."

"The following personal and work-site characteristics were significantly associated with the reporting of any symptom: female sex (odds ratio, 1.7; 95 percent confidence interval, 1.5 to 1.9), atopic illness (odds ratio 1.4; 95 percent confidence interval, 1.3 to 1.6), use of the computer for more than four hours each day (odds ratio, 1.3; 95 percent confidence interval, 1.1 to 1.5), working in an open area (odds ratio, 1.1; 95 percent confidence interval, 1.0 to 1.2), and younger age. These characteristics were also associated with the reporting of mucosal or systemic symptoms."

"In this study, increasing the supply of outdoor air in mechanically ventilated office buildings was not associated with either improved environmental ratings or a reduction in the number of symptoms reported by the participants. Key features of the design were that three randomized crossover trials of experimental manipulation of outdoor-air supply were conducted, during which the participants, who were unaware of the study intervention, completed standard questionnaires under different environmental conditions."

"The failure to detect a significant association between the level of ventilation and the number of symptoms reported may have occurred for several reasons. The participants may not have had the sick building syndrome."

"The buildings studied may not have been sick."

"The supply of outdoor air in the work sites may have been poorly estimated from measurements of carbon dioxide."

"The reduction in the supply of outdoor air may have been insufficient to produce symptoms."

"In this study, temperature, humidity, and air velocity varied significantly between work sites and contributed to differences between subjects in the number of symptoms reported."

"In conclusion, the supply of outdoor air was experimentally increased, from an average of 7 percent of the total air supply in the ventilation system and 30 ft^3 of outdoor air per minute per person at work sites to an average of 32 percent and 64 ft^3 of outdoor air per minute per person, respectively, and these increases were associated with significant changes in the concentrations of contaminants at different work sites. These changes were not associated with the participants' ratings of the environment or with the number of symptoms reported that were considered typical of the sick building syndrome. We believe that this research method can be used in further studies to establish a scientific basis for ventilation and contaminant standards to ensure the health and safety of the majority of North American workers."

[40] **"The Sick Building Syndrome In Office Buildings — A Breath of Fresh Air,"** K. Kreiss, ***The New England Journal of Medicine*** 328(12): 877-878, 1993

"The paper by Menzies and coworkers in this issue of the *Journal* brings fresh air to the largely untested hypothesis that the sick building syndrome is related to inadequate ventilation with outdoor air. The authors found that symptoms at work (not necessarily attributed to the building) and dissatisfaction with the environment in the building bore no relation to the rates of ventilation with outdoor air in buildings whose ventilation rates were considerably above not only the current standard but also the pre-energy crisis standard of 20 ft^3 (0.57 m^3) per minute per person. . . . As an experimental study, the work of Menzies et al. is the most robust of these efforts in rejecting the hypothesis that increasing ventilation with outdoor air above the current standard will eliminate the symptoms of the sick building syndrome. This study does not, however, address the more interesting question of whether increased ventilation with outdoor air can reduce the rate of symptoms in buildings with low rates of ventilation."

"If increased amounts of outdoor air do not reduce complaints about the quality of the air in buildings whose ventilation rates are already high, what else might cause the sick building syndrome? . . . High rates of building-related symptoms are common, but the rates vary substantially among buildings, implying that the causes may be preventable. The correlation between symptoms and measurable contaminants, such as

2046361374

Source: https://www.industrydocuments.ucsf.edu/docs/hqwl0000

formaldehyde, volatile organic compounds, ions, and particulates, is poor, although more work characterizing microenvironments within buildings may be helpful."

"At this point, we do not know the cause of the sick building syndrome, and the science to support prevention, correction, and the setting of standards is woefully undeveloped and unsupported in the United States. The problem requires multidisciplinary efforts in the experimental manipulation of buildings and the epidemiologic characterization of building-related measurements and occupant-related risk factors. The paper by Menzies et al. stands out as an illustration of sound experimental and epidemiologic methodology in the field of indoor-air quality."

"Altering the characteristics of a building that are responsible for the sick building syndrome is usually not under the control of clinicians or office workers, and the scope of public health action is limited in the absence of scientifically based standards. . . . To resolve building-related symptoms, practicing clinicians must enlist the efforts of public health agencies, epidemiologists, ventilation engineers, and building managers to understand specific buildings and the characteristics of building furnishings and ventilation systems that are associated with the well-being of a building's occupants."

2046361372

Source: https://www.industrydocuments.ucsf.edu/docs/hqwl0000